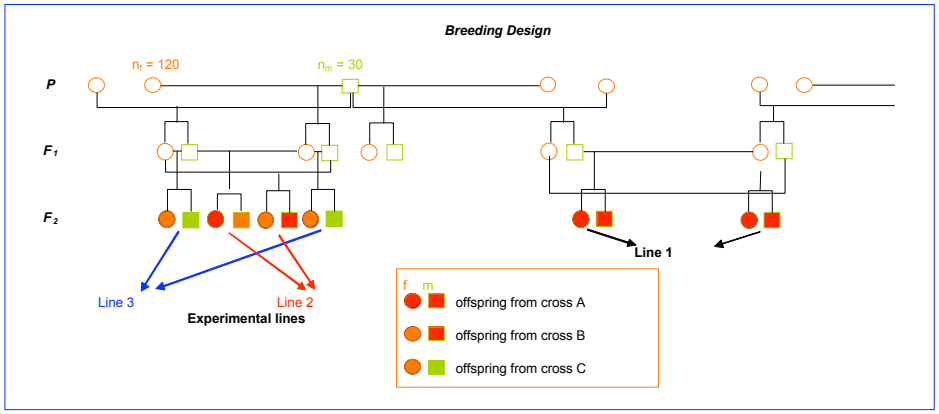
Genetic Variation in Disease Resistance of Chinook Salmon (*Oncorhynchus tshawytscha*) Exposed to Two Bacterial Pathogens



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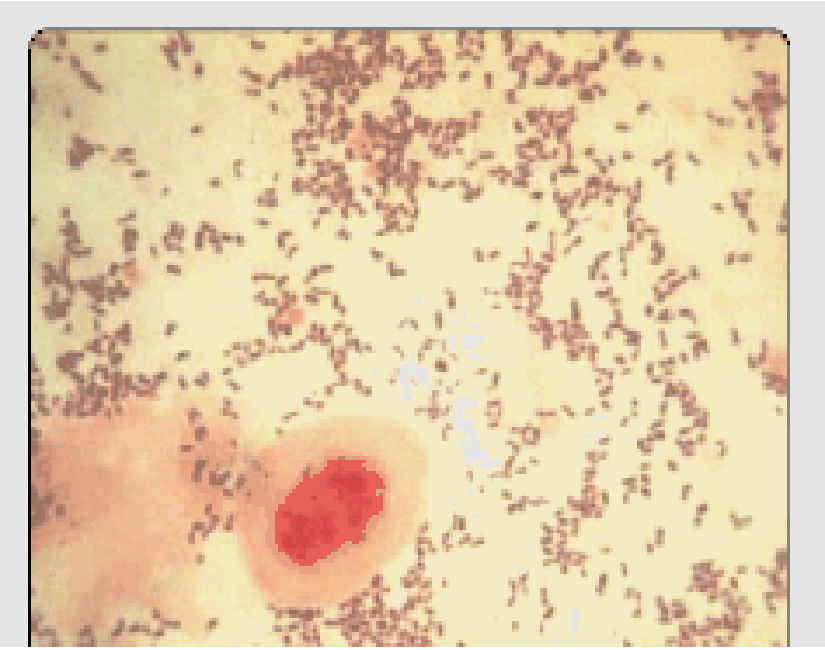
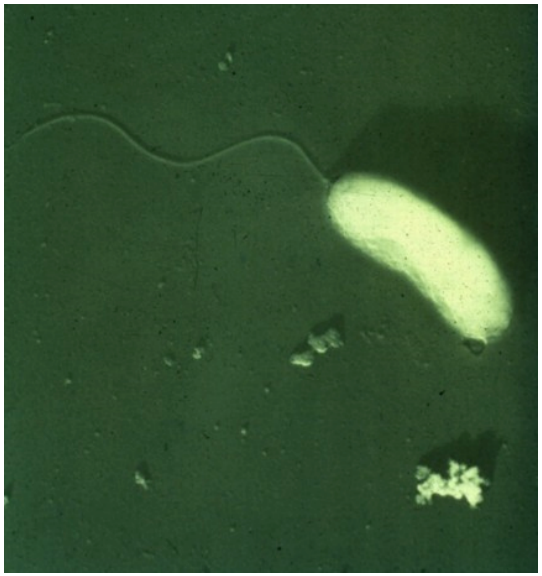
Study organisms



Renibacterium salmoninarum (FAO Finfish Diseases)



Listonella anguillarum (J. Crosa, OHSU)

Juvenile chinook salmon with BKD (NWFSC Microbiology)

Vibriosis hemorrhage (www.aquaculture.bz)

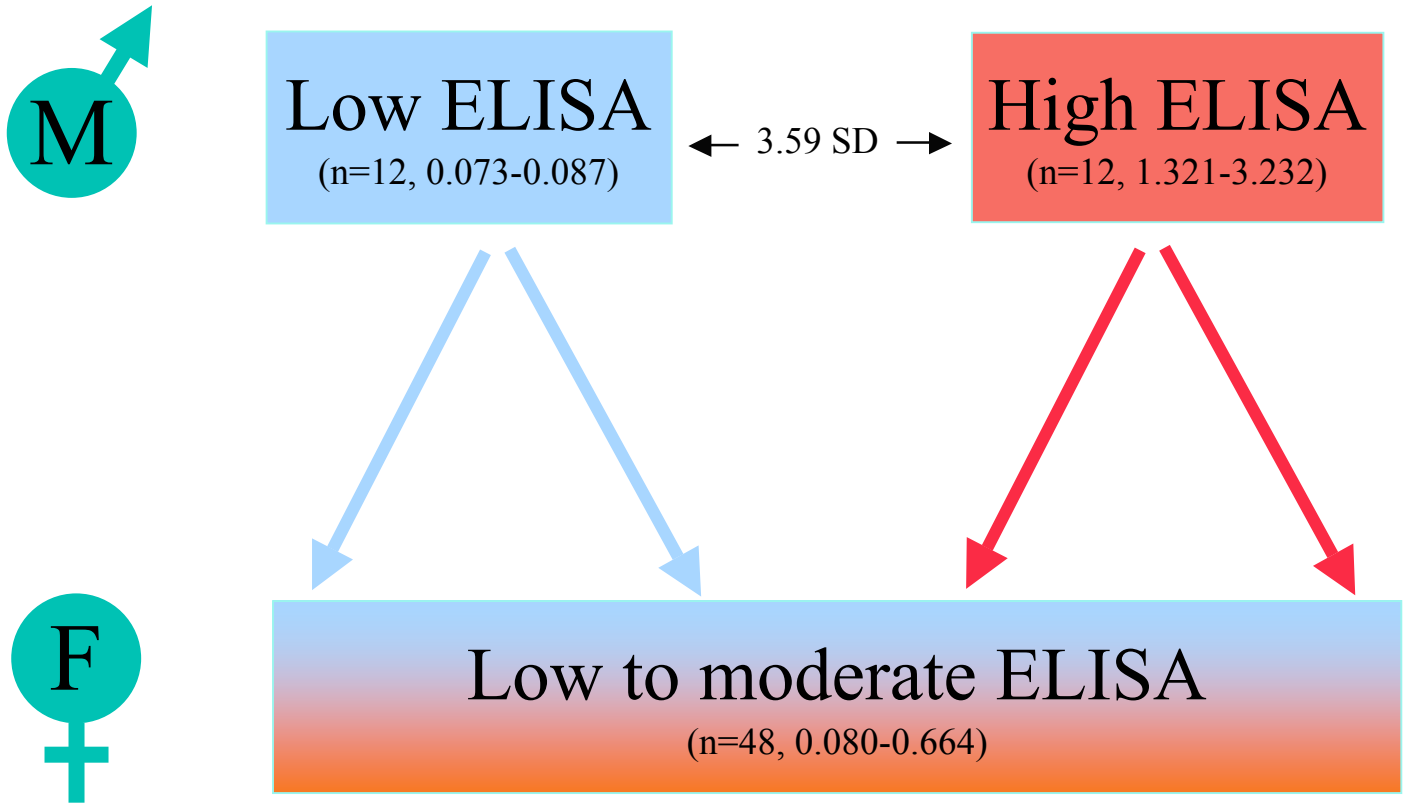
Background

- Bacterial kidney disease (BKD), caused by *Renibacterium salmoninarum*, is widespread in salmon hatcheries in the Pacific Northwest
- BKD is difficult to control because effective vaccines have not yet been developed; antibiotic therapy is only partially effective
- Oral chemotherapy of salmonids with erythromycin can reduce mortality but does not eliminate infections from all treated fish
- Many hatchery programs selectively cull progeny of adults that exhibit high antigen titers (ELISA) for *R. salmoninarum* to minimize risk of BKD outbreaks
  - Culling probably reduces likelihood of disease
  - Is culling likely to affect future disease resistance?

Study questions

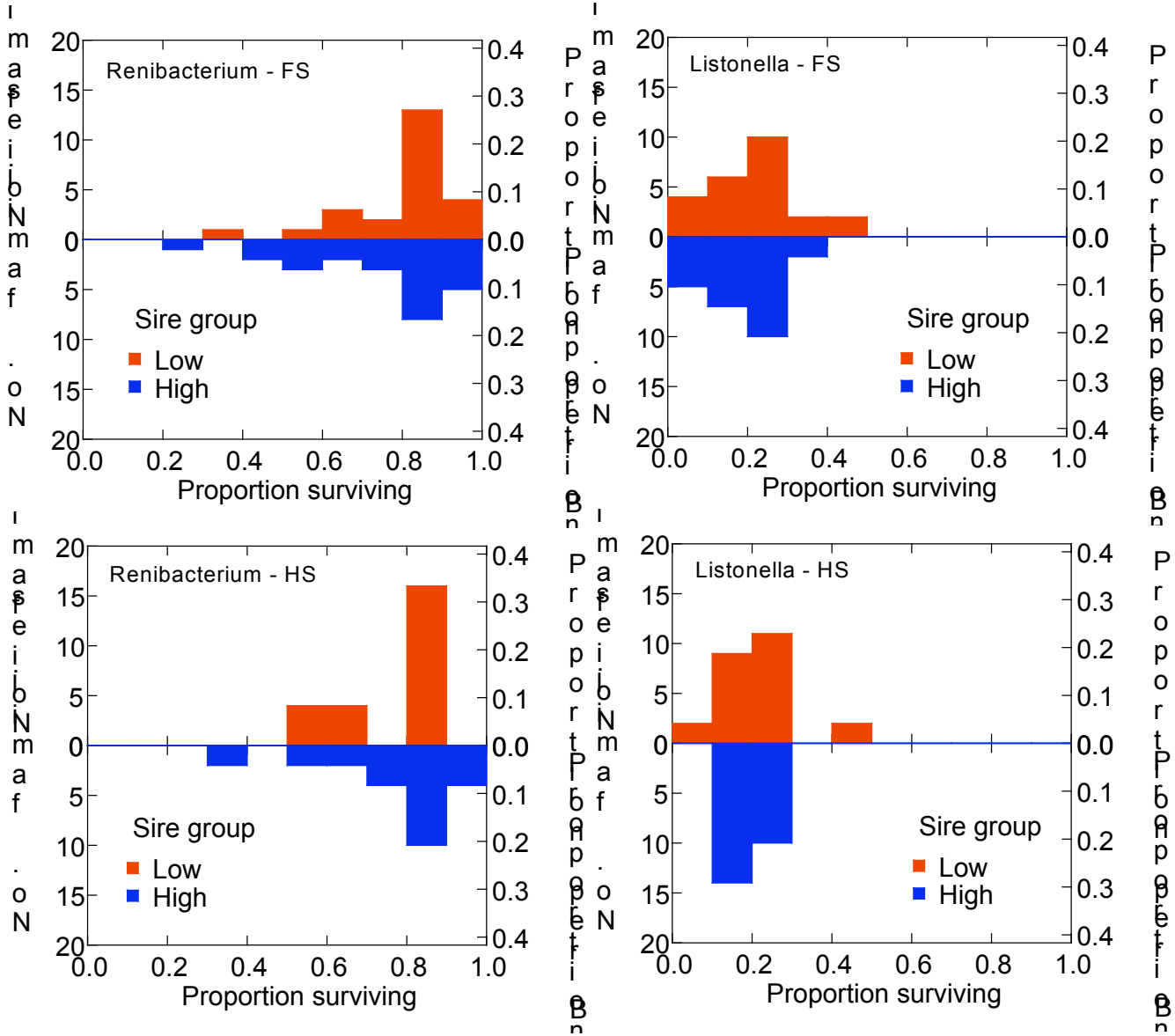
- Does the level of *R. salmoninarum* antigen, as measured by ELISA, in adult chinook salmon indicate the susceptibilities of their progeny to infection by *R. salmoninarum* or by *Listonella* (formerly *Vibrio*) *anguillarum*?
- What is the degree of genetic influence on variation in the two susceptibilities?
- Is there evidence that the two susceptibilities can evolve independently?
- What are the implications of the relationship between susceptibilities for salmon broodstock management and disease control?

Breeding design

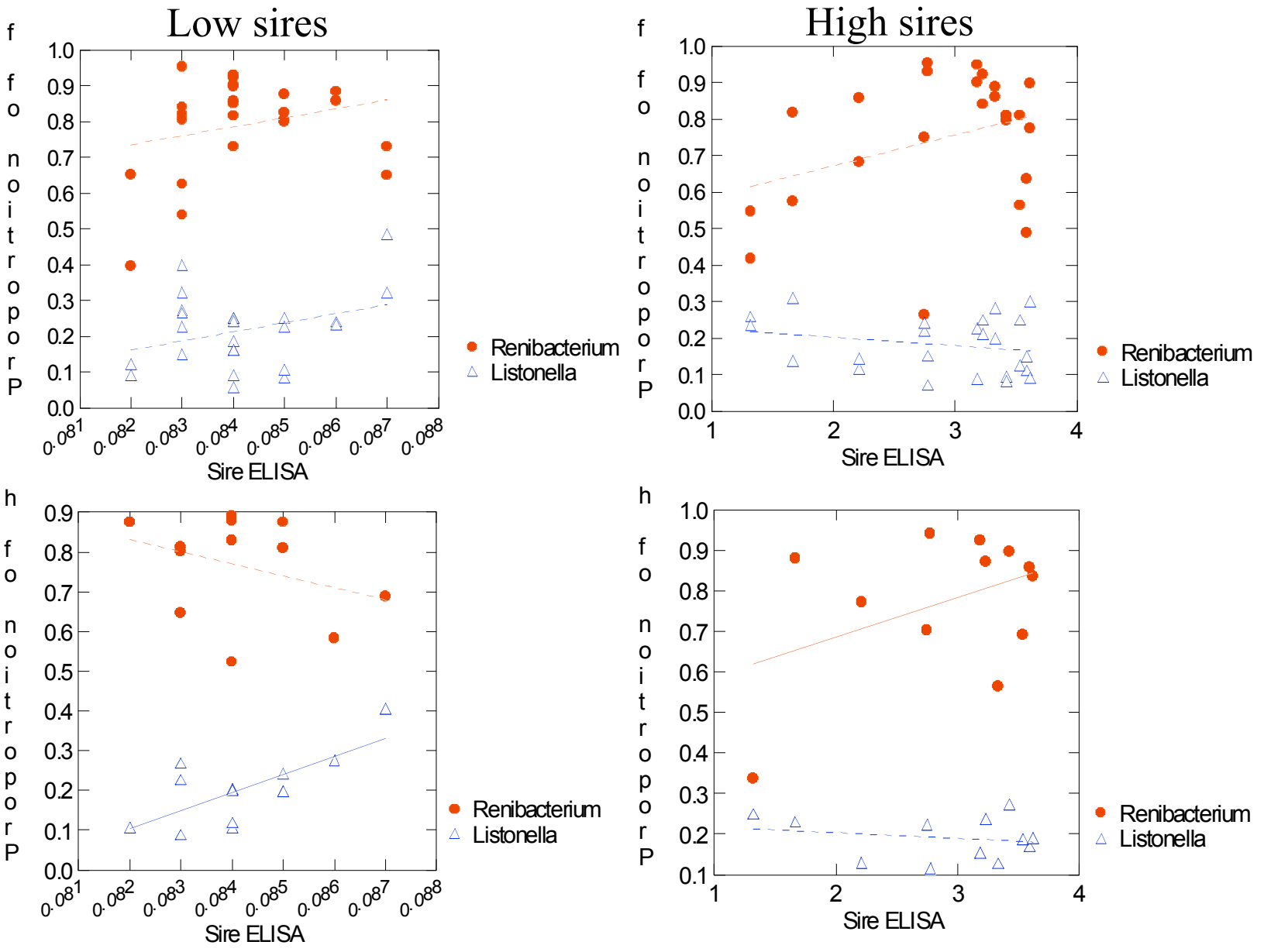


- A total of 48 families created from 24 males and 48 females (415 males and 84 females screened, 84 original families constructed)
- 3392 fish PIT tagged and phenotypes evaluated

Patterns among families in survival



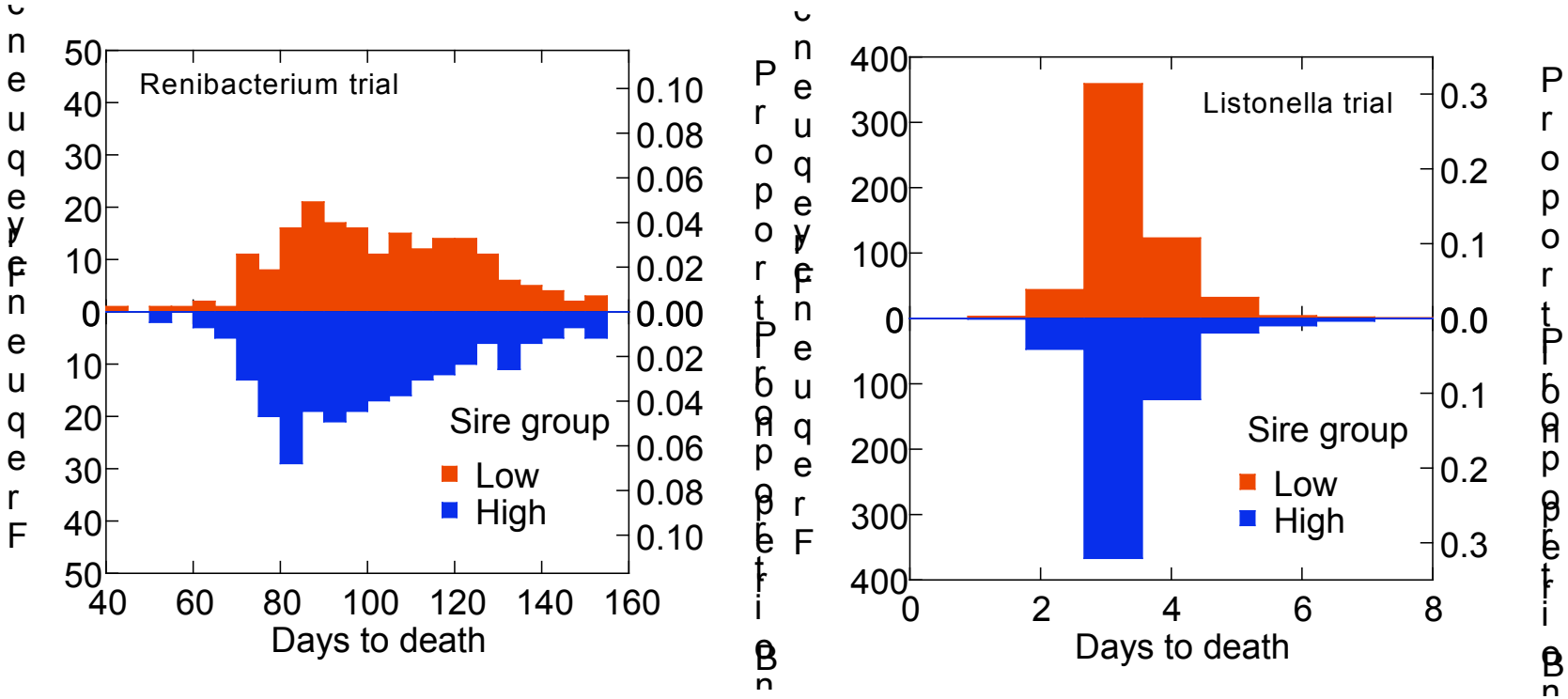
Proportion surviving vs sire ELISA



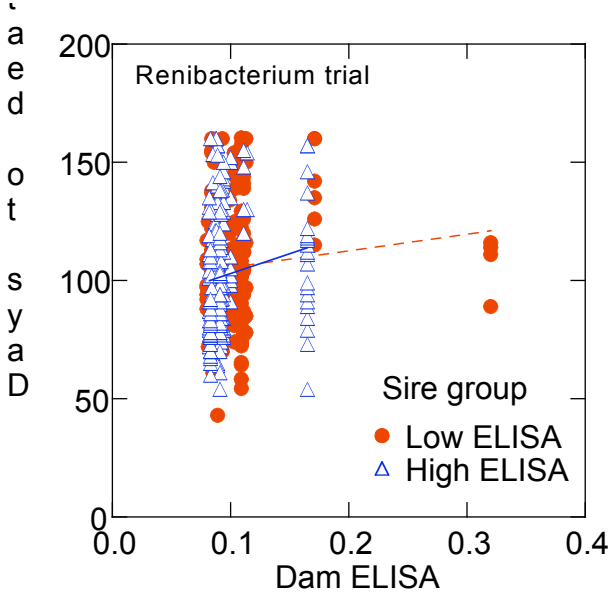
Survival

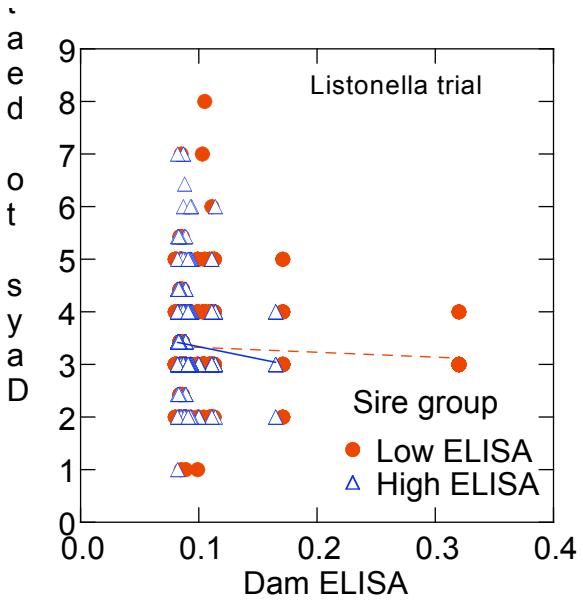
- In both pathogen trials, survival varied substantially among half-sib (but not full-sib) families, depending on the sire ELISA
- Proportion surviving in the *Renibacterium* trial was higher for progeny of sires with low ELISAs; proportions surviving in the *Listonella* trial was similar for progeny in both sire groups
- Proportion surviving in the *Renibacterium* trial increased with sire ELISA in the high sire group (may be artifact)
- Proportion surviving in the *Listonella* trial increased with sire ELISA in the low sire group
- Proportion surviving in either trial did not vary significantly with dam ELISA, regardless of sire ELISA

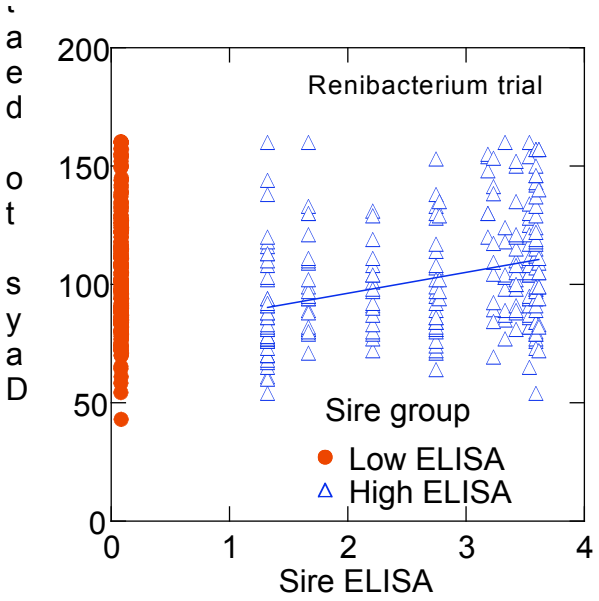
Temporal distributions of mortalities

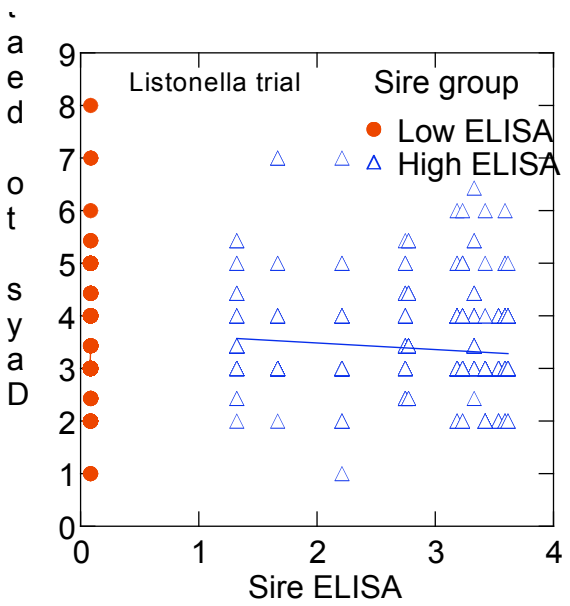


Patterns in days to death





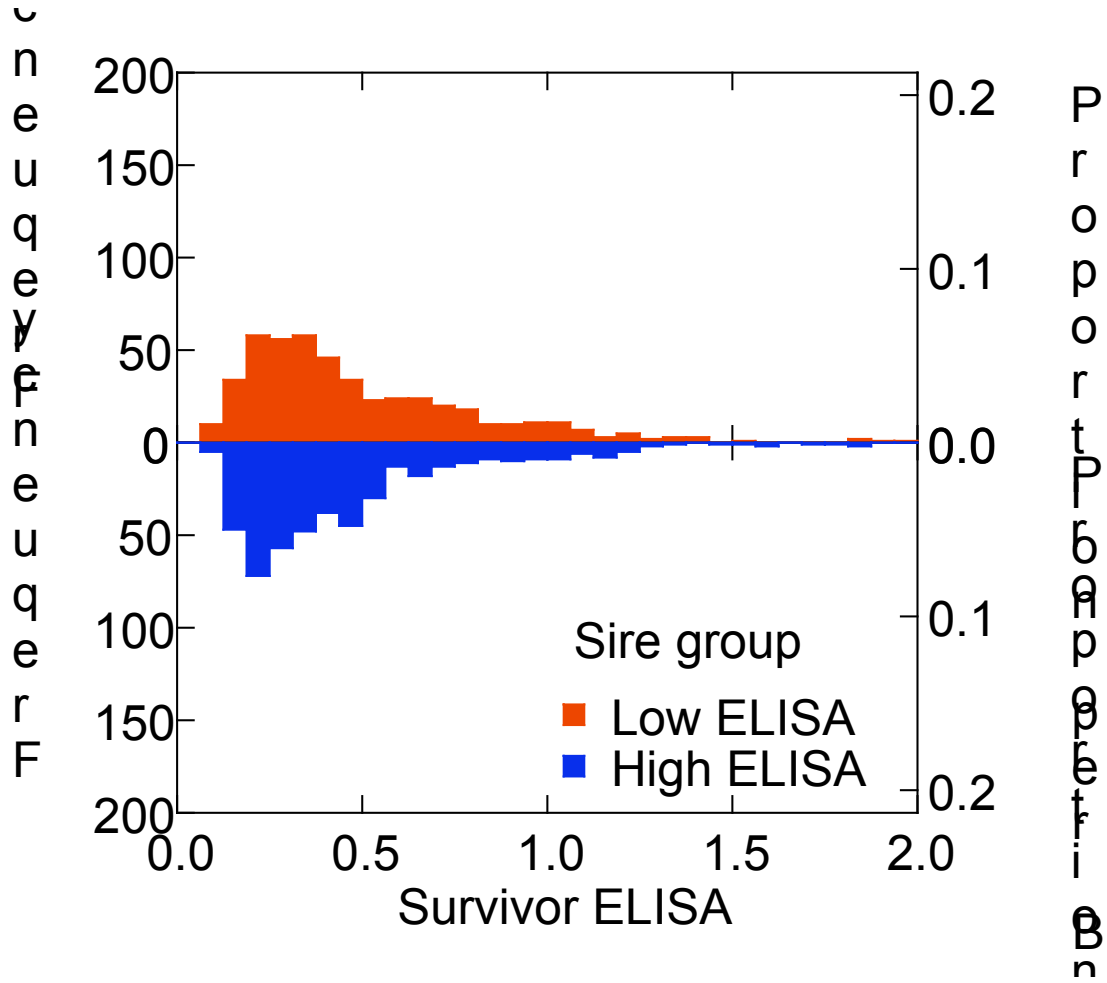




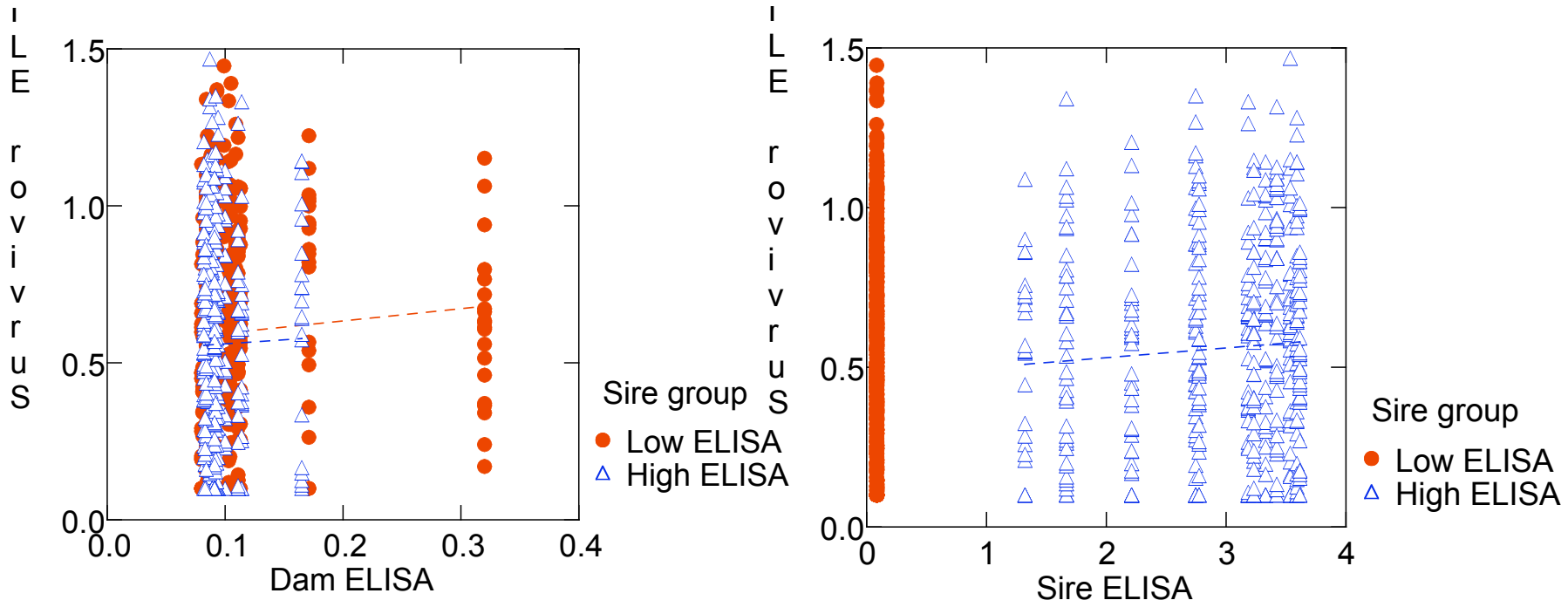
Days to death

- In both pathogen trials, days to death varied substantially among full- and half-sib families
- In both trials, mean days to death were similar in progeny of sires with low vs high ELISAs
- For progeny of sires with high ELISAs, days to death varied significantly with both sire and dam ELISA
  - Longevity increased with dam and sire ELISA in the *Renibacterium* trial
  - Longevity decreased with dam and sire ELISA in the *Listonella* trial

Survivor ELISA



Survivor and parent ELISAs



Survivor ELISAs

- ELISAs of survivors varied substantially among both half- and full-sib families
- Survivor ELISAs were higher in the low sire group, but did not vary detectably with either parental ELISA
- Survivors from families with higher survival rates tended to have lower ELISAs than those with lower survival rates, but the relationship was weak

Additional key results

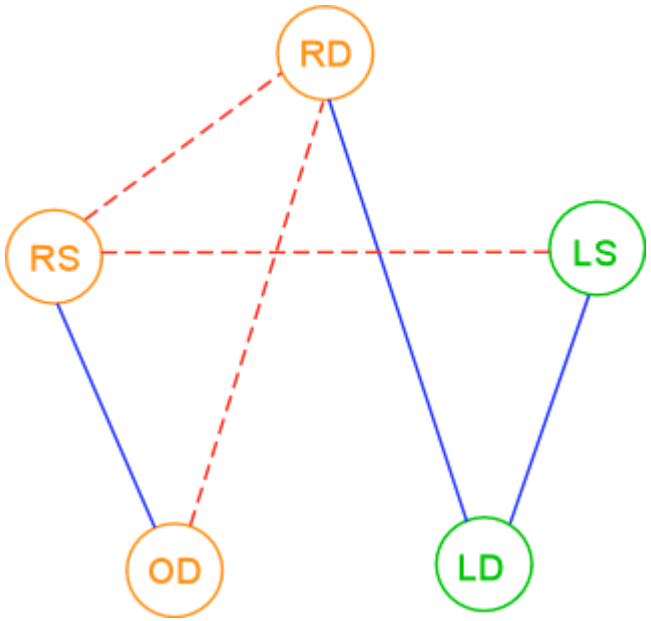
- Families with higher survival rates when challenged with *Renibacterium* had lower survival rates when challenged with *Listonella*
- In both challenges, mortalities in families with higher survival rates tended to die sooner
- Families with members that died sooner in the *Renibacterium* challenge had members that lived longer in the *Listonella* challenge

Trait variation and heritability

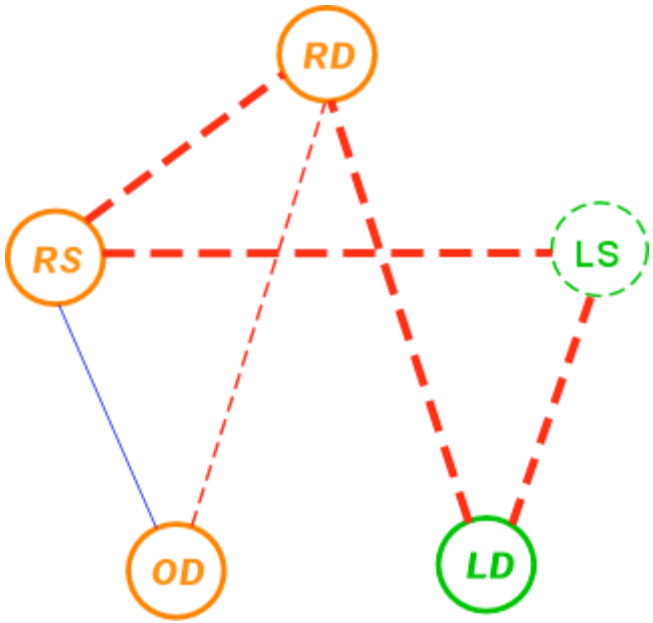
Trait	n	Mean	V_P	CV	V_A	h^2	SE(h^2)
RS	1962	0.771	0.176	54.466	0.042	**0.455**	0.101
RD	450	103.711	598.142	23.582	105.307	**0.176**	0.063
LS	1430	0.200	0.196	199.870	0.005	0.069	0.044
LD	1144	3.343	0.615	23.541	0.084	**0.137**	0.044
OD	942	0.578	0.110	57.281	0.027	**0.249**	0.069

Architecture of trait variation

Phenotypic



Genetic



Conclusions

- Survival on exposure to *Renibacterium* but not *Listonella* showed evidence of substantial genetic influence; longevity in both trials showed evidence of modest genetic influence
- Survival in the *Renibacterium* challenge varied with sire but not dam ELISA, was higher for progeny of sires with low ELISA, and increased with sire ELISA in the high sire group
- Survival in the *Listonella* trial increased with sire ELISA in the low sire group
- Longevity depended on parental ELISAs in the high but not low sire group
- In the *Renibacterium* trial, survivors from families with higher survival tended to have lower ELISAs, but no clear evidence that ELISAs are related to parental titers (or reflect variation in “resistance”)
- Inverse genetic relationships for several traits expressed in responses to the two pathogens suggests that antagonistic pleiotropy underlies the basis for the different responses

Implications

- Study results underscore the complexity of resistance of salmonids to bacterial pathogens and indicate the potential for rapid evolution of host resistance
- Results are consistent with a hypothesis that host responses to the two pathogens differ
- Study results provide no evidence that genetic variation in antigen load is linked to resistance to *Renibacterium*, as measured by survival
- Results suggest a genetic consequence of culling hatchery broodstock based on ELISA titers
- Among the potential longer-term outcomes of such BKD control practices is reduced resistance to *Listonella* as well as altered resistance to *Renibacterium*